# Section of Psychiatry

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## Paper

## The Families of Schizophrenic Patients

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My first study dealing with families of schizophrenic patients was confined to the mothers and to the mother-child relationship in schizophrenia. Employing the psychiatric interview, I investigated the mothers of 100 schizophrenic patients under 30 years of age, and the mothers of 20 neurotic patients and of 20 normal controls. The Rorschach test was administered to 92 of the mothers of schizophrenics and to all mothers in the control series. In a monograph (Alanen 1958) I classified the mothers I had studied into clinical categories on a unidimensional scale. The distribution that resulted is illustrated in Table 1.

I found that 12 of the mothers of schizophrenics had been manifestly psychotic and that a further 11 displayed unrealistic thought and behaviour patterns bordering on the psychotic. These mothers were assigned to the diagnostic categories A and B respectively. The most impressive finding, in my opinion, was the high frequency of mothers who were not included either in the psychotic or in the borderline group but were typified by a very accentuated constriction of affective life, poor self-control and an inability to feel themselves into the inner life of other people. Some of them were schizoid personalities; others were, above all, very aggressive and embittered; and, finally, some were very anxious and insecure, often with obsessional features. I separated these mothers from those whom I considered to be afflicted by 'ordinary-level' psychoneurotic symptomatology. In Table 1 the schizoid and other nonpsychotic but emotionally very disordered mothers are included in category C and the ordinarily psychoneurotic mothers in category D. The last category, E, includes the mothers who manifested only slight neurotic traits or were completely healthy.

*Table 1*
**Distribution of mothers of schizophrenics, neurotics and normal persons by clinical category** (Alanen 1958)

| | *Clinical category of mothers* | | | | | |
|---|---|---|---|---|---|---|
| *Series* | *A* | *B* | *C* | *D* | *A+B+C+D* | *E* |
| Typical schizophrenics | 7 | 5 | 27 | 9 | 48 | 6 |
| Schizophreniform psychotics | 5 | 4 | 12 | 12 | 33 | 8 |
| Schizophrenia plus organic disorder | — | 2 | 1 | — | 3 | 2 |
| Total schizophrenics | 12 | 11 | 40 | 21 | 84 | 16 |
| Neurotics | — | 1 | 1 | 7 | 9 | 11 |
| Normals | — | — | 1 | 5 | 6 | 14 |

For definition of clinical categories A–E *see* text

A larger proportion of the mothers of typically ('process') schizophrenic patients than of the mothers in the more benign schizophreniform psychosis group (diagnoses made according to Langfeldt 1953) fell within category C, which comprises the schizoid, aggressive and emotionally constricted mothers. I made a comparable observation when I tried to form a picture of the psychodynamic mother-child relationship in this study. Emotionally cold personalities with firmly dominating attitudes toward the child were definitely more frequent among mothers of typically schizophrenic patients than among those in the schizophreniform psychosis group, where warmer, more softly overprotective mother-child relationships were more common.

The psychoanalytic view of schizophrenia places a great emphasis on the disturbed early mother-child relationship. Although in my material this was often the case, it also seemed that the disturbing influence had not been uniformly concentrated in the patients' infancy or their mother relationships. More relevant appeared to be the frequent occurrence of continued disturbances, associated with a disordered mother-child relationship and liable to suppress

*Table 2*
**Distribution of parents and siblings of typical schizophrenic and neurotic patients by clinical category (Alanen *et al.* 1966)**

| | *Degree of disturbance category* | | | | | | |
|---|---|---|---|---|---|---|---|
| *Series* | *VI* | *V* | *IV* | *III* | *II* | *I* | *?* |
| Parents of schizophrenics | 4 | 12 | 26 | 12 | 1 | — | 5 |
| Fathers | 1 | 6 | 14 | 5 | — | — | 4 |
| Mothers | 3 | 6 | 12 | 7 | 1 | — | 1 |
| Parents of neurotics | — | 1 | 10 | 25 | 16 | 2 | 6 |
| Fathers | — | — | 6 | 10 | 8 | 2 | 4 |
| Mothers | — | 1 | 4 | 15 | 8 | — | 2 |
| Siblings of schizophrenics | 4 | 6 | 10 | 11 | 18 | — | — |
| Siblings of neurotics | — | — | 4 | 17 | 24 | 4 | — |

Degree of disturbance categories: VI = schizophrenia; V = other functional psychoses, borderline psychotic features; IV = schizoid, paranoid and cyclic character disorders, alcoholism, psychopathy, sexual perversions, very severe character neuroses; III = milder psychoneurotic symptoms or personalities; II = normal with mild disorder traits; I = normal without disorder traits

the child's activity, and in some cases the father appeared to be more pathogenic than the mother.

The second study, conducted together with a number of my colleagues, was planned to investigate, through psychiatric interviews and a psychological test battery, members of the families of 30 schizophrenic and 30 neurotic patients (Alanen *et al.* 1966). Fifteen male and 15 female patients were included in both groups, and only typical schizophrenics with 'primary' symptoms and typical cases of neuroses treated in psychiatric hospitals were admitted. The descriptive findings regarding the parents and siblings of our index subjects are illustrated in Table 2.

The earlier findings concerning the mothers of schizophrenics were confirmed. Table 2 also reveals, however, that disorders in the fathers were not less marked. Among the 55 parents of schizophrenics we had four who were schizophrenics themselves (category VI) – a proportion very compatible with the findings of most genetic studies. Of the 12 parents included in the next column (category V), five had psychotic-level paranoid symptoms, four displayed features suggestive of borderline schizophrenia, and three exhibited or had exhibited psychotic traits of an affective type. The most conspicuous feature about the distribution of the fathers and mothers of schizophrenics was their high frequency in category IV. Schizoid, paranoid and obsessional disorders formed a distinct majority in this group, and this was especially so for the mothers; about half the fathers in this group were alcoholics. Milder neurotic disturbances corresponding to category III were less frequent; but, as also appears from Table 2, they predominated in the group of parents of neurotic patients.

Among the siblings of schizophrenics the disturbances displayed greater variability than among their parents. There were four manifestly schizophrenic siblings and six who had borderline psychotic traits. Among the remainder, however, a wide variety of psychopathic and neurotic disorder patterns were found, though a considerable proportion of the siblings were regarded as normal. Neurotic disorders were comparatively common in the siblings of neurotics, but more than half of them were free from such disorders.

Findings of this kind suggest that, rather than being a specific and precisely circumscribed disease entity, schizophrenia should be regarded as the extreme degree of a more general tendency in these families toward developing psychic disorders of a particular kind. Genetic studies of illnesses that, in clinical practice, fall within the borderland between schizophrenia and other psychic disorders, as it were, point in the same direction (*see*, e.g. Shields 1968). Disorders other than schizophrenia were also of frequent occurrence in the families of schizophrenics according to the investigation conducted by Atkinson *et al.* (1968) in Newcastle; in this study no psychiatric diagnosis was recorded unless there was evidence that the relative had required psychiatric treatment or had experienced a major disruption of his social or working life as a result of illness or personality difficulties.

Such a view of schizophrenia does not, of course, imply anything concerning the nature of the causes of this disorder: these may be genetic, as well as associated with environmental factors. It is quite possible, indeed, that the disturbed relatives whose illness has not reached the stage of schizophrenia are suffering from the milder formes frustes of the same disorder, and that these also rest on a genetic basis. I would be inclined to think, however, that even if such a conception were accepted, our findings speak for polygenic rather than for specifically monogenic heredity. At this point I also wish to refer to the twin studies carried out by Tienari (1963, 1968) and Kringlen (1967) in the Scandinavian countries on the basis of population registers. Both found the strict concordance of monozygotic twins with respect to schizophrenia to be much lower than the figures reported in well-known previous studies (Kallmann 1946, Slater 1953).

I shall now try to give a brief account of some observations we made concerning the psychodynamics of the families of our schizophrenic patients.

Of 30 such families, 14 were regarded by us as schismatic and 7 as skewed in Lidz sense (Lidz *et al.* 1957). Six of the families had broken before the patients reached the age of 12; thus only two or three families could be considered normal or well integrated as we characterized them. We also classified our families into two groups, chaotic and rigid, according to the nature of general atmosphere typical of them. Ten of the families were chaotic and 11 were rigid; six families had

both chaotic and rigid features, and 3 were atypical. General incoherence was characteristic of the chaotic families, and in the worst cases the family atmosphere was coloured with psychotic thought patterns and attitudes, which also typified the parents' relationships with their children. On the other hand, the atmosphere in rigid families was emotionally impoverished, schematized and unbending, and the parents (or one parent) usually related to their children in a very possessive or restrictive way, tinged with strict expectations. Although the children in both types of families undoubtedly had experienced severe emotional frustrations, the parents in these families could not be characterized as rejective towards their children; quite to the contrary, in many cases the children were quite important to the parents, providing them with compensation for disappointments encountered in their own childhood and in their marriages. The situation in such cases can be described by stating that children had remained the only object into which the parent could still direct his or her wishes for emotional satisfaction. Distinct differences were met between the roles of various siblings in many families, depending on both genetically based and psychological factors.

How could such disorders of the family environment be related to the pathogenesis of schizophrenia? The personality development of every individual is influenced by two sets of factors: his hereditary endowment and the experiences associated with his environment during growth. Neither set can eliminate the influence of the other: both are invariably present and contribute to the genesis of disorders in personality development, and I think that the genesis of schizophrenia is not an exception to this rule.

The family, and a child's parents in particular, occupy a key position in the process transmitting people's earliest and most fundamental experiences from one generation to the next. Parents influence their children's development both genetically and extragenetically. The nature of the extragenetic influences may be illuminated by dividing them into two broad groups as follows: parents have a part to play as their children's earliest emotional objects on the one hand, and as the persons with whom the children identify and from whom they learn on the other.

The family environments from which schizophrenics come are usually disturbed in both respects. In particular, they have lacked healthy models of identification from whom to learn inward adjustments and adjustments to external environments. Instead, many schizophrenics have learned in their childhood homes attitudes that markedly differ from those of other people in their environment, and even psychotic attitudes and modes of behaviour, which they may employ in critical developmental phases as models conducive to illness. Another fact clearly in evidence is the pathogenic impact of ties of dependence. Where the parent has been accustomed to regard the child as a complement of his or her own personality, through which to satisfy unfulfilled wishes and onto which to direct projected fears and prohibitions, it will be difficult for the child to find a way to well-integrated independence. (In this connexion, *see* also the concept of 'double-bind' described by Bateson *et al.* 1956.)

I agree with Manfred Bleuler (1968) who stated that 'if you know the life history of a schizophrenic in intimate detail, you always find close temporal connections between environmental circumstances and the psychotic evolution'. Nevertheless, although factors precipitating the onset of psychosis can be discovered, the dynamically most important part is not played by such factors but, instead, by the personality that has remained weak and prone to regression. I think that in schizophrenics the process of neutralization, necessary for the development of the defensive functions of the ego, has remained defective and the underlying infantile instinctive drivings chaotically uncontrolled. The factors responsible for this state of affairs may include a lack of stable and socially useful identification patterns, as well as difficulty in reconciling needs and emotions associated with the early object relationships.

The practical result of our studies has been family psychotherapy of schizophrenic patients. As I see it, family therapy in its various forms should play an important role in the treatment of schizophrenics. The most effective form of therapy is usually that in which the patient and his parents – and sometimes some of the siblings – attend the therapeutic sessions conjointly. We have found that such a therapeutic situation in itself provides the schizophrenic patient with a powerful stimulus to leave his autistic inner world. After an initial contact, families of schizophrenics frequently show a willingness to come for therapy, either because one or both of the parents are afflicted by severe anxiety, or because of the symbiotic relationship they may have with the patient. This often makes it possible to help not only the patient but also the other family members. From the point of view of primary prevention, there would be reason to enlarge the scope of family therapy so as to include married schizophrenic patients' own families and by developing the after-care of patients along lines compatible with the family-therapeutic concept. This is all the more important in view of the con-

stantly increasing number of cases in which schizophrenic patients are now able, by using the growing psychiatric outpatient services, to live at home with their families.

In conclusion, I would like to emphasize again that psychodynamically oriented family researchers – or at least a majority of them – do not deny the importance of genetic factors in the development of schizophrenia. Rather, we are inclined to consider the genetic and environmental factors as a complementary chain of etiological causes. Neither do I believe that psychological and physiological approaches to the study of schizophrenia are mutually exclusive or irreconcilable. I expect that an approach to the problems of schizophrenia which seeks to combine and integrate results of studies representing different orientations and methods will prove most fruitful, and that psychodynamic study of the family and of family therapy will have an increasing part to play in the future.

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## DISCUSSION

**Dr Eliot Slater** (*Institute of Psychiatry, London*)

When Professor Alanen's famous book 'The Mothers of Schizophrenic Patients' appeared in 1958, it caused something that can only be called a world sensation. It remains today one of the great clinical studies of the family background of schizophrenics, by a subtle and sensitive observer and a stimulating and original thinker. Professor Alanen has reminded us of some of the main findings of the report of 1958, and he has given us a fascinating account of how his work and that of his colleagues has progressed since.

From a different standpoint, I hope to consider whether the genetic factors which Professor Alanen concedes, and the environmental factors which the geneticist concedes, can be brought into a meaningful synthesis.

Professor Alanen particularly studied the mothers of schizophrenics, but their fathers also have not escaped his attention; he thinks that in many cases they are just as pathogenic as the mothers, or even more so. This view is confirmed by the observations of Kallmann in Germany and of Bleuler in Switzerland, both of whom found that it was just as dangerous for the child to have a schizophrenic father as a schizophrenic mother. The point is an important one, since the child socially and emotionally is so much closer to the mother than the father, though on the genetic dimension at an equal distance from both.

Professor Alanen has found much more psychopathology in the mothers of schizophrenics than in the mothers of neurotic and of normal control groups: 63 out of the 100 mothers of schizophrenics showed gross psychopathology of grades A, B and C, as against 10% of the mothers of neurotics and 5% of the mothers of normals. Penetrating more deeply, he finds the relationship of parent to child more deviant and potentially dangerous in the schizophrenic group than the others.

Potentially dangerous, yes. But has the relationship been shown to be actually pathogenic? And pathogenic in a specifically schizophrenogenic way? We must remember that the genetic hypothesis leads one to expect constitutional deviations, schizoid personalities and the like, in the parents of schizophrenics; and abnormal personalities can be expected to behave in an abnormal way. But is this abnormal behaviour the direct cause of the child's illness?

Professor Alanen has pointed out that parents may be very upset indeed when they see one of their children go mad. One may think that, under such a terrible blow, they might become aggressive and embittered, or anxious and insecure. They might perhaps, when put on the spot, give a resentful or defensive or slanted picture of the precursors of that catastrophe. I cannot feel that Professor Alanen's observations will bring much comfort and cheer to that person whom, if he exists, I would call the panpsycho-etiologist.

Let the geneticist offer a model for your consideration. We envisage a group of disorders, much akin clinically, of which some of the rarer syndromes are exogenously caused. A typical member of the group is determined by a single major autosomal gene, manifesting with reduced penetrance in the heterozygote. The condition shows itself very rarely under the age of 15, but after that is liable to cause a remitting and relapsing illness in which mental symptoms are prominent in 80% of cases – the symptoms we call schizophrenic, paranoia, depression, irritability, restlessness, hallucinations, confusion. The attacks may be precipitated by menstruation,

pregnancy, infections and various drugs. Repeated attacks are likely to leave the patient with some degree of permanent damage. The disorder to which I refer is porphyria, particularly acute intermittent porphyria. But I could just as well have been referring to schizophrenia. Would it be fair to take the one as a working model to help us understand the other?

What, then, are the lessons we should draw from our contrasting views? Where should we devote our main efforts, along genetic-biochemical or along social-psychological lines? Practical consequences of immense importance hang upon our choice.

If we suppose a major gene to be the specific cause of some serious illness, we are tempted to think of the predisposed individual as being predestined. So he is, as long as we fail to recognize that this is the case. Once we confirm the genetic determination, we can discover the nature of the metabolic disorder; and then, inserting our chemical instrument into the 'inevitable' chain of cause and effect, we break it off short. So it has been with phenylketonuria. Now that we know this condition is caused by a recessive gene, causing defective hydroxylation of phenylalanine, we can diagnose the abnormality in early infancy; we can put the child on an appropriate diet; and we can rear him to normality.

But what happens if we discover that the main cause of a serious condition lies in our psychological make-up and our social life? Recent history gives the answer. We have discovered that the smoking of cigarettes is currently the main cause of lung cancer, but society finds it impossible to make any effective use of this knowledge. Every year some 20,000 men and 4,000 women are dying of cancer of the trachea, bronchus and lung; and more cigarettes are being smoked every year. Rushing round the country in motor cars at high speeds costs us some 6–7,000 deaths a year, and many times more that number of disabled and maimed. There is hardly a soul who wishes to take any step to stop it. Our addiction to alcohol causes us large-scale problems in accidents and chronic disease. Prophylaxis remains impotent.

I pray that we shall find a specific genetic cause for schizophrenia, for then we may hope that something effective will be done in its prevention. If we find the causes lie in the bemused attitudes of only too human parents towards their children, we can be sure that the problem of prevention will be insoluble as long as the human family persists.

The following paper was also read:

**Family Variables in the Etiology of Schizophrenia**
Dr D A Stephens

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*Meeting May 21 1969*
*at the Royal Victoria Infirmary,*
*Newcastle upon Tyne*

# Computer Methods in Psychiatry: Diagnosis and Classification

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**Miss Elizabeth Barraclough**
(*University Computing Laboratory, Newcastle upon Tyne*)

## The Uses and Abuses of Computers
[*Summary*]

The conventional use of computers for performing large arithmetic calculations has become so routine that there is a danger that results from computers are treated as gospel. Anyone using a computer should keep remembering that it is merely an obedient moron; results produced should always be assessed for credibility. In medicine this check could well be whether the answers are clinically as well as numerically sensible.

The expansion of computer use in the future will almost certainly be in non-numerical applications. Large stores are becoming economical and facilities for handling text are increasing. Because of these developments the way is now open to provide assistance to doctors in many areas, such as aids to diagnosis, rapid access to stored patients' records and the use of case histories to improve diagnostic techniques. The initiative for these developments must come from the medical community as only they know the problems.